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Gum Formation With Special Reference to Cankers and Decays of Woody Plants

BY

B. B. HIGGINS

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GUM FORMATION WITH SPECIAL REFERENCE TO CANKERS AND DECAYS OF WOODY PLANTS

By B. B. Higgins.

In a recent publication on "Plum Wilt" it was suggested that gummosis was probably brought about by action of an enzyme whose formation from a zymogen might be induced by many forms of excitation. Several attempts have since been made to isolate a pectin dissolving enzyme from gumming wood, but always without success. In view of the fact that such an enzyme is usually found in freshly formed gum it seemed probable that the failure to demonstrate its presence in the wood was due to faulty technique; but as this fault was never overcome it became desirable to study more exactly the reaction of some gum producing plants to certain stimuli, to thus eliminate, if possible, some of the various theories and explanations of gum formation which have been advanced during the past few years.

Such data, if properly recorded and studied, would allow a comparison of this phenomenon with well known enzyme reactions and would determine its correlation with ordinary growth activity in the plant. For the purpose of such study, various experiments in artificial production of gummosis under partially controlled conditions were run and these experiments with resulting conclusions seem worth recording at this time.

Relation of Moisture to Gummosis.

The form of gummosis most frequently studied has been that following traumatism, yet little light has been thrown on the factors active in causing the transformation of cell membranes around wounds.

The most obvious physiological disturbance in such wounded tissue is the changed moisture relations. The removal or destruction of the partially impervious covering of cork or cuticle allows greatly increased loss of moisture from the underlying tissue. Prael (18) and later Ruhland (20) found that when the cut ends of twigs were protected from contact with air by covering with wax, glass tubes, etc., gum was not formed. On this basis Ruhland attributed gum formation to action of atmospheric oxygen on the exposed tissue. He failed to consider drying of the exposed tissue as a factor.

In order to determine this and related points at question a series of experiments was started during January, 1916. Twigs of various species of *Prunus* were brought to the laboratory, cut under water into five to seven inch pieces and set in bottles containing water sufficient to cover the lower cut surface. Preliminary tests showed that twigs of the Mahaleb cherry (*P. Mahaleb.L*) were most satisfactory for the work and they were used in all subsequent experiments except where otherwise noted. It was noted also that cutting the twigs under water or exposed in the air made no perceptible difference, but to insure exact uniformity all were cut under water.

On January 15th vigorous one-year-old twigs of Mahaleb cherry were cut and placed in four bottles containing about two inches of water which was replenished when necessary. All four were set in an incubator running at a temperature of 27°C. One bottle was closed with a ground glass stopper while the other three were left open. Gum began to appear in small pearly droplets on the exposed ends of the twigs in open bottles on the fifth day and examination at the end of eleven days showed results as given in Table 1.

TABLE NO. I.

Bottle Number	No. of Twigs	Gum on Cut Surface	Gum Under Bark	Callus	Per Cent Gumming	Bud Growth
1	22	0	0	0	0	0
2	18	7	7	0	77.7	Good
3	39	25	8	8	84.6	Good
4	40	9	27	0	90	Good

The fact should be noted that numbers 2 and 4 were comparatively shallow open vessels and most of the twigs projected considerably above the top while 1 and 3 were bottles 6½ inches high and even in the open bottle No 3 the twigs did not project. This is of interest in connection with the behavior of the twigs in the individual bottles. In No. 3 the majority of the twigs formed gum on the cut surface; a few (eight) of the larger pieces showed no gum on the surface but pockets of gum had accumulated under the bark lower down on the twigs, while the shorter pieces whose cut surface was well down within the bottle formed no gum whatever but abundant callus growth over the cambial region of the surface. In Nos. 2 and 4 the results were reversed. Comparatively few twigs formed gum on the cut surface and the greater number formed gum under the bark at a greater or less distance from the cut end—in some cases very near and in others as much as half an inch distant. Some of the

small twigs with poorly suberized bark dried up and died without forming gum. In the few cases where callus was formed it soon broke down forming gum. Similar results have been noted in all tests made during the past two winters.

The results seem to indicate that slight drying or evaporation from the wounded surface was the factor necessary to initiate gum formation, and the lack of such drying prevented its formation on the twigs of No. 1. It had been noticed, however, that there was considerable accumulation of gas in the closed vessels, sufficient in some cases to burst the vessel, and because of the fact that there was no bud or callus growth in the closed vessels it was thought probable that some deleterious gas formed by growth of microorganisms prevented growth and gum formation.

To determine this point a set of four bottles was set up and circulation of air provided as follows:

No. 1—Air drawn through two bottles of water, one outside and one inside incubator.

No. 2—Air drawn through one bottle of water outside incubator.

No. 3—Air drawn through two tubes of $CaCl_2$.

No. 4—Check. Left open.

The first three were attached to aspirator bottles and regulated so that air was drawn through each bottle at as nearly uniform rate as possible (about twenty liters each day).

After ten days the twigs were removed and examined carefully with results as follows:

TABLE NO. II.

Bottle	No. of Twigs	Gum on Cut Surface	Gum Under Bark	Callus	Per cent Gumming	Bud Growth
1	34	0	0	Slight	0	0
2	27	0	0	Slight	0	0
3	40	4	0	Abundant on neaarly all	10	Fair
4	18	13	3	Much dried up later	88.9	Good

These results seem to show conclusively that the gum formation is initiated by the slight drying, and they also indicate that bud and callus growth will not start in a saturated atmosphere. In No. 3 only four twigs nearest the intake tube, where air from $CaCl_2$ tubes first struck, formed gum while nearly all formed abundant callus. A very slight callus could be seen on only a few

twigs in Nos. 1 and 2 and the buds had not opened at all; but after standing exposed to the air for a few days twigs in all began to put out leaves, showing that they were still viable.

Relation of Temperature.

A great many tests of the relation of temperature to enzyme activity have shown marked acceleration of action with a small rise in incubation temperature up to an optimum which varies with different enzymes. The optimum temperature of pectinase of *Bacillus carotovorus* and other soft rot bacteria was reported by Jones as lying between 40° and 45°C. Above this temperature the action was retarded and finally stopped altogether at 51°C.

In the experiments on the relation of moisture a considerable variation was noticed in the length of time necessary for gum to appear on the twigs held at various temperatures but because of the difficulty of controlling other factors an exact test of this variation was not feasible. An attempt was made, however, to determine approximately the maximum and minimum temperatures of visible gum formation.

On January 15th eight bottles were filled from one lot of twigs. Four were incubated at 55°C and the other four at 10-15°C. The twigs at the higher temperature showed gum on the second day and after three days the entire cut surface of the twigs was covered with dark brown gum, and the wood to the depth of one-half to one centimeter below the surface, was filled with gum. To bring about the necessary drying in the low-temperature set, a dish of $CaCl_2$ was placed just above the bottles and was replaced with fresh $CaCl_2$, as the first began to go into solution. During the first two weeks the temperature was held near 10° but warmer days sent the temperature up slightly and at the end of another week gum had appeared in clear droplets on the end of three twigs and all had begun to develop callus. The buds were beginning to open at this time.

These results with certain observations in the field to be noted later indicate that the minimum temperature at which perceptible gum formation occurs is near 10°C. The low temperature experiment just described was not repeated, but further attempts were made to discover the maximum. Thirty-eight twigs were run at 60° with results similar to first. Dark brown gum had appeared on the surface of the twigs after thirty hours. At the end of three days the twigs were plainly dead, the bark and cambium was darkened but no gum could be found more than one-half to one centimeter below exposed wood of cut surface.

In another series twigs of Mahaleb cherry, wild cherry (*Prunus serotina*), plum (Japanese varieties), and persimmon were run at a temperature near 60°C with results in the case of all *Prunus* species very similar to preceding. With the persim-

mon twigs very little gum was visible on the cut surface, but when split lengthwise a very distinct area of gum filled tissue was found usually extending from about two to four millimeters below the surface to two and a half to three centimeters down the twig. The ducts as well as the parenchyma cells were mostly filled with gum and the wood when dried became very hard and resembled ebony.

In another series half a lot of peach, plum, and cherry twigs were boiled for fifteen minutes and then incubated at 80°C. The rest were run at the same temperature without previous boiling. In other tests the twigs were boiled sixty minutes and incubated at 90-100°C. In all these tests some gum was formed. Usually none was found in the bark or pith but in the older wood gum formed through a greater area of the twig than in twigs not boiled. This was probably due to the fact that the increased permeability, due to boiling, of the corky layer of bark allowed drying to occur over a greater area than in unboiled twigs. The formation of gum in twigs after they had been boiled seemed at first thought to indicate that the process is not due to enzyme action; but the relation of enzymes to heat is too imperfectly understood to justify such conclusions. A great many cases of enzymes withstanding high temperatures under the protection of various substances in solutions have been reported. Schmidt* reported that trypsin withstood boiling
in the presence of gelatin, and Mellanby and Wooley* accorded similar protective action to acid reaction of solution containing pepsin. Many other reports of such protective action may be found in recent works on enzymes. We know also that many living organisms can withstand boiling for considerable lengths of time, probably on account of protective action of covering membrane, and the physiologically inactive state of the protoplasm. Also those who have attempted to sterilize dead infected wood are familiar with the protective action of the wood on the contained organisms. Doubtless it would protect enzymes in the same way. The bark and pith, being composed of living cells with thinner walls in close contact, would naturally be a much better conductor of heat than the woody tissue, and naturally would be the place where life processes would first be stopped, which corresponds very well with the above results.

Another feature which must not be overlooked is the possibility that we are dealing not with the enzyme but with a zymogen. Very little is known of the properties of the substance or substances which generate enzymes, but we have some evidence that they are more resistant to deleterious agents than are the enzymes themselves.

This may also be applied to the reaction at low temperatures.

*Original papers not seen. cf. Bayliss, ref. 2, p. 97.

Were a pectin dissolving enzyme in a free state present in the twigs action would probably occur at temperatures below 10°C, though not necessarily sufficient to produce visible gumming; but the reaction by which the pectinase is formed from its mother substance may require a higher temperature.

Relation of Chemical Poisons.

A great many experiments in inducing gum formation by action of certain chemicals have been conducted by Sorauer[2], Beijerinck[4], Butler[6], Floyd[7] and others; and a long list of chemicals have been found effective for this purpose. From the published information on the subject, however, no definite conclusion can be formed as to the exact relation of the various chemicals to the plant tissue and to the process of gum formation, because usually the time and place of formation has not been closely observed.

In order to study more closely the action of chemicals on the plant tissues as well as the time, place and nature of gum formation three chemicals (viz. $HgCl_2$, $CuSO_4$, and $(NH_4)_2SO_4$) were selected from the list of those reported as inducing gumming, and were used in several series of experiments. On August 5th five to eight branches each of peach, plum, and cherry (*P.serotina*) were treated with each of these chemicals. The bark of the twig was slit, a small crystal of the chemical inserted and the wound covered with grafting wax. After forty-eight hours gum had begun to break through the bark near the wounds receiving the $HgCl_2$ and $CuSO_4$. The bark of the branches was killed to a distance of six to twenty-four inches immediately above the wound, and in most cases, to about one inch below. At the end of six days some of the branches were almost entirely dead. The poisonous chemical had been carried by diffusion and the movement of water to the tip of each branch, the wood as well as the bark directly above the wound was dead, and the young tip of the axis was usually killed. The leaves had dropped from many branches not killed outright. When sectioned it was found that the wood was killed to a greater distance above and below the wound than was the bark. This would naturally be expected to occur because of the more rapid diffusion through the wood vessels. The lateral limits of the dead wood were sharply delimited even to the unaided eye by a darkening of the tissue. This darkening was due to a dense deposit of gum in all elements of wood around the outer limits of the poisoned tissue (see figure 5). Gum was not found in any appreciable quantity within the dead tissue. Occasionally a little was deposited in tissues made up largely of woody fibers or wood parenchyma, as for example in the bundle sheath. Where the poison passed through this sheath to the pith the latter was invariably killed and the limiting gum deposit was formed in the sheath and primary wood beyond.

Even when this sheath tissue was included within the dead area some gum was usually found as minute droplets over the pits in the walls. The youngest wood was broken down. Gum was formed in pockets in the cambial region, this being the gum which broke through the bark.

At the end of seventeen days no gum had appeared around the wounds receiving ammonium sulphate. The grafting wax was then removed and the wounds examined more carefully. All had begun to heal but in the wounds on the plum a small mass of gum was found. No killing of the tissues attributable to poisoning was evident and no gum had formed in the peach or cherry. The gum formation in the plum was evidently due to osmotic disturbance rather than to poisonous action of the ammonium sulphate.

The results with mercuric chloride and copper sulphate indicate clearly that a concentration of solution of these chemicals beyond a certain point prevents gum formation in the tissue, and that as the solution becomes more dilute through diffusion they, in some way, cause the liquifaction or transformation of certain components of the cell membrane. The observations show that this did not occur where the concentration was sufficient to kill the tissue outright. It seemed that one should therefore be able to make up solutions of sufficient concentration to cause gum formation without killing the tissue directly, if the solution could be properly applied, which possibility was demonstrated by the followng experiment.

Solutions of mercuric chloride were made up in concentrations of 1-500, 1-1000, 1-2000, 1-5000, 1-10000, 1-100,000. 1-500,000, 1-1,-000,000, and 1-10,000,000. Two hundred cubic centimeters of each solution were placed in a wide mouth bottle and numbered consecutively, beginning with the most concentrated, 1 to 9. In another bottle (No. 10) was placed 200 c. c. of tap water as a check. On September 27th vigorous peach branches of that season's growth were cut, placed quickly in a beaker of water and brought to the laboratory. From these the twigs were cut under water and divided as nearly equally as possible on the basis of amount of wood and of leaves, between the bottles, which were then set in a sunny spot in the laboratory. As the solution in each bottle was used up more of the same concentration was added.

After forty-eight hours the lower leaves and bark of the twigs in Nos. 1 and 2 had begun to die and the next day all in these two bottles were entirely dead. The leaves wilted and dried on the twigs and the bark turned brown. Drops of brown liquid which on examination was found to be gum, exuded from the bark. On the fourth day the twigs in Nos. 3 and 4 had begun to die and on the sixth day some twigs in No. 5 were dead. The twigs were killed much more slowly than in the first two, but in

exactly the same manner. The leaves wilted and dried still clinging to the twigs and great numbers of gum droplets oozed from the bark in some cases before the outer bark itself was killed. When sectioned and examined under the microscope the twigs in all cases showed considerable gum in the bark especially in and around bast areas. None could be found in the wood of the first two. In fact, the wood of these twigs seemed to be very well fixed. In Nos. 3 and 5 some gum could be found in areas where ducts were not abundant but none could be found in ducts or in cells adjoining them.

On the eighth day the leaves began to drop from the twigs in No. 6 and had nearly all dropped by the next day. The leaves dropped while still green and turgid. They had apparently been separated from the stem by an abscission layer as occurs in normal leaf fall, and as had previously been noted in twigs on poisoned branches some distance from the point of application. No gum appeared outside the bark except in a few cases near the surface of the solution where it broke through green bark from the cambial region; but gum was found quite abundant in the wood especially along the leaf traces. On the eighth day the leaves dropped from twigs in No. 7 in the same way. The twigs in Nos. 8 and 10 remained normal in appearance for several days but finally some began to turn yellow and drop their leaves, and in No. 9 the twigs were killed outright about the tenth day. In all three there was considerable fungus growth on the cut ends of the twigs. This growth was especially abundant in No. 9 where the dilute (1-10,000,000) mercuric chloride seemed to stimulate the fungus growth. In these cases, however, the symptoms were quite different from that produced by the more concentrated solutions where no fungus growth occurred. Many of the twigs in Nos. 8 and 10 remained normal in appearance until thrown out at the end of two weeks.

No doubt much of the mercuric chloride was absorbed by the non-living (cell walls, etc.) material in the twigs, and there was also doubtless some change in concentration in the twigs due to loss of moisture by transpiration. Also no account was taken of the amount of moisture already in the twigs; so one cannot say from these results just what concentration kills the tissue, or just what concentration is necessary to cause gum formation or leaf fall, but the method and results are sufficiently exact to show that the two latter phenomena are produced by a concentration somewhat lower than the killing concentration. The results also suggest an analogy between leaf fall or the formation of the abscission layer* and gum formation.

Assuming that gum formation is an enzymatic process, we would expect it to be active under conditions which favor growth and

*As described for other members of this genus by Lee[14].

general enzyme activity of the plant, though not necessarily concomitant with it. For instance, we know that the enzymes active in growth and carbohydrate formation in the plant may become inactive and the plant go into a resting state presumably because of accumulation of products of their activity, while conditions of temperature and moisture favor continued activity. If now the pectinase be formed, or, as Beijerinck[3] assumes, released through the retaining plasmatic membrane, we should have gum forming when the plant is in a complete resting condition in so far as growth is concerned. On the other hand, Butler's conclusions* would make enzyme activity in this connection much more difficult to explain.

In studying this phase of the problem, experiments in artificial gum production were carried out during the resting period and also during different phases of the growing period.

January 5, 1916, a young healthy Red June plum was inoculated with a culture of *Lasiodiplodia triflorae* at four points. The bark was slit, a small amount of fungus mycelium inserted, and the wound then wrapped with paraffined cloth. The temperature remained unusually high, and when the tree was examined three weeks later, gum was found in all the wounds and one mass of gum had broken through the bark about two inches above one inoculation. Gum was also found abundant in the infected wood. There was no sign of growth activity until about the middle of March when the flower buds began to open.

There was, of course, a possibility that a pectin dissolving enzyme was secreted by the fungus, so in a similar experiment the following winter a chemical poison was used instead of the fungus.

On November 18, 1916, a few days after the first freeze, a small amount of $CuSO_4$ was inserted under the bark at one point on each of seven large well developed water sprouts on young peach trees in the manner previously described and the wounds covered with grafting wax. After a few days the twigs arising from the sprouts near the point where the poison was inserted had begun to die as evidenced by discoloration and wilting of bark. The temperature remained comparatively low, however, and no gum appeared until the twenty-seventh day (Dec. 7th), when, after four days of higher temperature (maximum 22°C and minimum 5.5°C, with an average for the four days of 20°C maximum and 10°C minimum) when gum began to break through the bark. A microscopical examination showed well developed gum pockets around the cambial region of affected twigs and a heavy gum deposit in wood bordering that killed by the poison. To this date much more of the tissue had been killed by the poi-

*cf. ref. 6, p. 132, "For gummosis to develop, two conditions must be simultaneously fulfilled. The cambium must be actively laying down new tissue elements, and a superabundance of water must be present."

son than in like sprouts treated earlier in the fall. In some cases large sprouts one-half inch thick were entirely dead, which emphasizes the protective action of the gum in stopping the diffusion of the poison.

Several similar experiments were performed at various seasons, but one other performed during the early growing period will suffice to show the general behavior of poisons.

April 26, 1917, four vigorously growing peach water sprouts about one centimeter thick were treated with $CuSO_4$ as in the previous experiments and at the same time six twigs of the previous years growth were given exactly similar treatment with as nearly as possible equal amounts of the copper sulphate in each case. Four days later a narrow strip of tissue immediately above the point of application on the young water sprouts had been killed to within four to six inches of the tip (the entire tip of one sprout was killed), and side branches and leaves along this area were dead. The upper twelve inches of the sprout was drawn almost into a semicircle by the dying and shrinking of tissue along one side. Gum had broken through the bark along the border of the dead bark, a layer of tissue densely filled with gum surrounded the dead wood, and a row of gum pockets extended entirely around the stem in many places, the leaves had been shed from the older parts of the sprouts and their side branches, and in many cases a small droplet of gum was found on the leaf scar. The young immature leaves did not fall. Among the inoculated branches a great deal more tissue was killed in the older than in the younger sprouts, but otherwise the results were similar. In neither case did there appear to be any further killing of the tissue after the fourth day. The greater destruction of tissue in the older branches can probably be explained by the more rapid diffusion of the poison through the well developed ducts. In the young water-sprouts the conducting tissue was not well developed, and also much of the copper sulphate was probably absorbed by the proteids which are of course more abundant in young wood.

While the flow of gum from the older branches was greater than from similar branches treated during the winter it was certainly no greater than during the warmer fall months after all cambial activity had ceased. The gum which breaks through the bark is formed partly from the walls of certain cutical elements but principally from the undifferentiated cells of the cambial region. One might therefore expect gum flow to be much more copious during rapid growth; but it seems that the walls of the undifferentiated cambial tissue are during the summer months thickened by a deposit of pectic substances which augment the gum mass.

That an actively growing condition of the plant is not essential for gum formation is shown positively by these results.

Gum in Woody Tissue of Other Plants.

While examining some twig cankers of oak a gum-like substance was noted in the wood elements around the infected tissue, and examination of other diseased woody plants showed the presence of a similar substance.

In studying the early literature of the subject it was found that gum had been demonstrated in a great variety of plants by Boehm[5], Gaunersdorfer[9], Frank[8], Temme[25], Molisch[18] and Prael[19].

Boehm, in studying the function of wood ducts, found that young wood of *Ailanthus*, *Amorpha*, *Catalpa* and *Robinia*, was easily permeable to air; but the older wood was practically impermeable to air. This he found due to tyloses in the vessels of *Catalpa* and *Robinia* and to gum in those vessels of *Ailanthus* and *Amorpha*. He found this condition not only in heart wood, but also in twig stumps in the border of dead tissue.

Gaunersdorfer studied the formation and properties of "Kernholz," as he termed gum-filled or tylose-filled tissue whether produced by age or by wounding, in Syringia vulgaris and also in species of *Aesculus*, *Prunus*, *Cytisus* and *Diospyros*. He found the production of such wood induced by both causes to be identical. He also was led to believe that the gum formed in such tissue resulted not from cell wall but from transformation of the stored starch and other cell contents.

Frank and his students, Temme and Prael, extended these studies considerably and from their results conclude that gum is formed as an end product of plant metabolism and not as a metamorphosis or disintegration product of the cell membrane. In this Frank followed the theory first announced by Prilleaux (see Molisch[18]). He applied the term "Schutzholz" (shieldwood) to gum-filled discolored wood around wounds, to distinguish it from similar heartwood "Kernholz," forming the core or center of most woody dicotyledonous stems.

Temme found in the heartwood of *Gleditschia triacanthus*, *Quercus pedunculatus*, *Pyrus malus* and *Prunus avimum*, a brownish brittle deposit which he found to be gum exactly similar in all respects to that formed in shield wood. It swelled but did not dissolve in cold or hot water, was insoluble in alcohol, ether, alkalies, sulphuric acid, nitric acid, and aqua regia. By nitric acid and potassium chloride it is broken down forming mucic acid and oxalic acid.

Prael made a comparative study of the sap wood, the heart wood, and the shield wood or "Schutzholz" of twenty-eight species of woody plants distributed among twelve different families, though all three phases were not obtained for study in all cases. He found that in sap wood the cell walls are colorless and the lumen contain only starch, calcium oxalate, and other normal cell

constituents; but in heart and shield wood the cell walls were darkened and the vessels were filled with (1) gum, with (2) resinous substances, or with (3) tyloses. Among his conclusions the following are of special interest:

(1) Shield-wood formed about wounds appears constantly similar to heart wood of the same species.

(2) The distinctive appearance of the heart of colored woods is caused by a distinctive coloring matter which apparently is formed in the cell contents and on the death of the protoplast passes into the cell membrane, probably combining chemically with the lignin. However, the idea that the membrane merely takes it up as soluble substance must not be entirely overlooked.

(3) Air tight coverings over wounds prevents or at least retards shield wood formation.

Molisch, though primarily studying tylose formation and function made many observation on gum formation over a wide range of species. He noted that the vessel gum, following pollution with lignin, often gives all lignin reactions, and cell walls not previously liquified often in the neighborhood of wounds, suffer chemical change and give all lignin reactions. He found that the intercellular spaces between the parenchyma cells were often filled with gum, giving the tissue the appearance of collenchyma.

While a great many other workers studied "gummosis" in *Prunus* and *Citrus* they dwelt usually with the more obvious symptoms, the appearance of gum pockets in the cambial region and of gum flowing from wounds or crevices in the bark. This work has been reviewed quite fully in the recent papers on the subject (see Butler[6]). The general occurrence of gum in wounded tissue of plants seems to have been quite generally overlooked during recent years and much information which might have extended our knowledge and understanding of gum formation in general has no doubt been lost. For instance the "humus compound," references to which are found in the recent plant disease literature is doubtless identical with the gum of these authors and is formed by the host tissue instead of by the parasite as conjectured by Von Schrenck[22]. Also a careful study of the "slimes" associated with some bacterial disease would probably show similar origin.

My attention was first called to the general occurrence of gum while examining cankers on twigs of the scarlet oak. A substance was found in the lumen of the wood parenchyma and fiber cells as well as in the vessels, which had all appearances of gum of peach and plum wood except that in the oak it gave tannin reactions. Since tannin was very abundant in the tissue it might reasonably be expected to be included with other cell contents in the gum mass. Later it was found that older gum in the ducts

often did not react with ferrous suphate. Examination of other cankered and diseased pants both woody and herbaceous showed the presence of a similar substance in the various elements of conducting tissue. A more thorough study of these plants was therefore planned, to learn if gum formation in these species was subject to the same influences as in species of *Prunus*.

To this end diseased and wounded parts were studied in the fresh condition. In most cases also material was killed in chrom-acetic (Schaefer's Solution) solution or in absolute alcohol, treated with hydrofluoric acid, and embedded in celloidin. It was found that gum about fresh wounds was largely dissolved, but in cankers and older wounds little perceptible loss occurred in most cases. For instance from peach and apple twigs killed six days after wounding with poisonous chemicals practically all the gum was dissolved and lost, whereas similar twigs from the same lot killed twenty-two days after wounding retained a large part of the gum. Comparisons were made with various reagents and stains which left no doubt of the general similarity of the deposited substances in the various plants studied.

For staining microtome sections of the embedded material ruthenium red-methyl green combination was found to be far the most satisfactory, though Boemer's haematoxylen gave very good results. With the former stain the sections were treated about thirty minutes with a mixture of 25 c. c. HCl and 75 c. c. 95% alcohol. They were then washed with a mixture of strong aqueous solution of ammonia mixed with equal parts of 95% alcohol, and afterward washed thoroughly with water. The sections were then covered with a dilute aqueous solution of ruthenium red for several hours (usually twelve to twenty-four) washed with water and then covered with a dilute aqueous solution of methyl green for ten to twenty minutes. This stain was then poured off, and the sections were washed in water, run up through the alcohols, and destained with Eieleschamer's clearing solution. This destaining is very important and often requires ten to twenty-four hours to give the best results. It has the advantage that one can examine the sections at intervals and see just when the best results are attained. The celloiden was then dissolved away in clove oil which was then replaced with cedar oil and from this the sections were mounted in balsam. Long treatment in the clearing solution reduces the solubility of the celloiden in clove oil. In many cases, with rather fragile tissue, the sections were mounted without removal of the celloiden, which gave very satisfactory mounts for most purposes.

The middle lamellae stained bright red while the secondary lamellae stained green. The gum showed greater or less attraction for one or the other stain, varying apparently with some physical (or possibly chemical) change in the gum. In the ducts, the gum was usually stained some shade of red though often a

portion of that in a single duct would stain red and the other portion green. The behavior in this respect was so intricate that no definite idea as to causes could be formulated. All diseased tissue took the methyl green much stronger than the healthy living tissue, but this should probably be attributed to physical change rather than to more intense liquifaction as Molisch has suggested.

Apple.

September 4, 1916, three healthy twigs of young apple tree were treated with copper sulphate as previously described; three others were treated with mercuric chloride. After six days the bark above the wounds had turned black and died to a distance of three to six inches; and sections showed that the wood was killed more extensively. Some twigs of each lot were removed and examined more carefully. It was found that gum had been deposited abundantly in the bark and also a dense deposit had been formed in the wood bordering the dead portion. The wood parenchyma cell as well as the vessels were filled with this deposit.

On the twenty-second day after treatment more careful examination was made and material was killed in absolute alcohol and run up for microtome sectioning. The poison had not spread appreciably in the bark since the sixth day and had apparently not done so in the wood. Little or no gum could be found within the dead area but a dense deposit had formed around the dead tissue (see figure 00) laterally as well as above and below. At this time the gum appeared to be insoluble in water but some was dissolved out during the process of killing, desilification, and washing. In the bark the dead tissue had been cut off by a cork layer.

Spraeropsis canker. During the late summer of 1916 several twig cankers produced by *Shacropsis malorum* were found in the Station orchard on one and two year old twigs. These cankers showed a dense layer of gum around the infected tissue, but little gum was found within the infected region. That present in this area was mostly confined to wood parenchyma and fiber cells; but in the surrounding tissues all elements were densely filled (fig. 6). Where this thick tissue included the pith gum had formed in all pitted conducting cells. In many cases gum was found in the angles between the ordinary parenchyma cells.

In the bark gum was found in the bast cells. The infected dead tissue had been cut off from the living by a layer of cork cells. No gum pockets were found in the cambial region.

Hesler[11] has recently made a very careful histological study of *Sphaeropsis* canker of apples. He noted the fact that a "gum-

like substance'' was formed around the infected area and also mentioned that this substance was largely confined to the wood parenchyma cells in the tissue infected with the fungus mycelium.

Phyllosticta Canker. The apple blotch fungus *Phyllosticta solitaria* Ell & E has been abundant throughout this region during the past two years and ample opportunity has been afforded for studying the cankers on both young and old twigs—in young cankers and those almost overgrown.

The fungus mycelium is confined almost entirely to the bark especially if the wood is farily well matured before infection occurs. In this case a dense deposit of gum is formed in the young wood of the cambial region, entrance of the fungus is thus barred and no gum is formed in the underlying wood. Where infection occurs on very young tissue the stimulus for gum foundation is communicated to the deeper lying wood tissue and gum deposits may extend to the bundle sheath and pith.

In the bark the behavior is very similar to that noted in *Sphaeropsis* canker as concerns the formation of a cork layer around the infected area; but reinfection of bark beyond this cork layer occurs quite frequently. This is in turn cut off and after awhile partly separates, at this layer, from the healthy bark, producing the scaly appearance of old cankers produced by this fungus.

Fire Blight. Early stages of fire blight were not examined, but the border between the healthy and the diseased tissue of twigs were examined sometime after death.

This border tissue was found densely filled with gum. The gum deposit extended down through the center of the twig some distance (variable) below the upper limit of the living bark and cambium. This, however, gave no definite clue as to the stimulus causing gum formation. The bacteria travel faster through the well developed vessels in the center of the twig. In case the bacteria secrete a poison we might then expect to find the gum extending further down in this region. If, as suggested by Bachmann, the bacteria do not secrete an injurious poison within the host tissue, then the stimulus may probably be attributed to drying of the tissue after death, which would also act to a greater depth in the center of the twig.

A ring of cork cells in the bark separates the healthy from the diseased tissue.

Insect Injury. Several cases of injury to the growing tips of young twigs by insects were studied. One of the twig tips had been killed apparently by the sting of some insect, and in two the tip was killed by the twig borer. In all, the twig had died for some distance below the point of injury, and a layer of gum

filled tissue separated the dead from the living portion. The deposit was very dense, filling all elements of the wood.

Decay. On old apple trees, fire blight often kills small twigs or fruit spurs on the large branches and passing down into the body of the branch produces cankers which finally give entrance to decay organisms. A large fifteen year old branch of a Siberian crab apple tree, which had been thus infected was removed and studied. It was impossible to identify the organism or organisms causing decay but a form of *Pleuratus ostreatus* Jacq. was found on a nearby decaying tree.

At the point of entrance at the original canker a large hole had been formed and all the inner wood had decayed, leaving only a thin shell next the bark. The decay extended only about six inches above and about ten inches below this original entrance point. The cavity was lined with a varying amount of dead partly decayed wood; and between this dead wood and the healthy outer tissue was a thin layer of black wood (fig. 2), which was densely filled with gum. It was evident that the decay organism had broken through this gum barrier and spread the infection a litte further at one or two points. Possibly water standing in the bottom of the cavity kept the gum soft or partially dissolved it, allowing the organism to progress more rapidly here than above.

Pear.

September 16, 1916, six pear twigs were treated with mercuric chloride and six with copper sulphate. No difference could be seen in the action of the two chemicals. The bark was killed for only a short distance around the wood, though the leaves dropped from the entire length of some twigs. The wood was blackened considerably further than was the bark. The cell walls of this region were discolored and the wood parenchyma cells were filled with a dark gummy substance; but very little gum could be found in the vessels. The experiment with copper sulphate was repeated with similar results.

Blight. Some study of pear blight in twigs was made; but the development of the disease seems to be so unusual and complex that no definite conclusion as to the relation between host and parasite or the relation of the bacteria to gum formation in the twigs could be drawn. This seems to be certain, however, that at the time the bark first turns black there is usually very little gum visible. Very soon, however, gum begins to appear in the ducts and wood cells, and sometimes gum appears in the bark. The gummy exudate from the bark of such twigs is probably formed from the pectins of the host tissue rather than the bacterial substance. On March 3, 1916, ten flower clusters of a supposedly resistant variety were sprayed with a pure culture of

B. Amolovorus. Eight of the ten soon showed signs of infection and the youngest growth was killed; but infection in no case passed back into the mature wood of the previous season's growth. Young twigs of this variety were inoculated at another time and showed similar results. Thirty days after inoculation, when it became evident that the disease was no longer spreading, some of the fruit spurs and young twigs were examined. Near the border of living and dead tissue the young wood had been split and pried into segments by masses of gum. The crevices extended down into the living wood (fig. 7). The gum in this region was blackened by contaminating substances. The wood vessels were filled with normal brownish colored gum for some distance into the living tissue.

The question as to whether this partially resistant variety forms gum more readily or abundantly than those more susceptible to blight was not determined. It seems quite probable, however, that some such physiological or structural peculiarity prevents the spread of the bacteria through the tissue of this pear as well as of apples. In both cases flower infection seems to occur as readily as in the less resistant varieties, but only a little damage is produced by each infection in the former. The question as to whether gum formation is, in this disease, initiated by the bacteria or merely by the drying which follows their activity in the twig is of primary importance in determining the relation of gum to resistance; but this can be determined only by carefully conducted experiments.

Persimmon (Diospyros sp.) Young.

On October 4, 1916, three twigs of a Japanese persimmon (*D. Kaka*) were wounded and treated with copper sulphate. Eight days later the wood was killed and blackened throughout almost the entire cross section of stems at points where poison was applied. Little gum had formed here, but when the twig was cut eight to twelve inches below this point, considerable quantities of clear, almost colorless, gum exuded in small droplets from the ducts. The gum finally hardened and turned brown on the surface as well as in the tissues. In November the experiment was repeated with similar results.

The persimmon is subject to a very serious Black Spot and Streak Disease, the cause of which is not yet known, in which the leaf tissue turns black in spots of various sizes and the infection passing down apparently through the leaf petiole, produces black streaks down through the wood and bark of the twig. The disease is very bad on certain varieties of Japanese persimmons, often killing large branches. Some of these diseased twigs were examined for the presence of gum. Considerable quantities of a black gummy substance were found in the ducts and paren-

chyma cells of the blackened area. This substance reacted like gum and seemed to be merely gum contaminated with a coloring matter which also stains the cell walls of dying tissues.

Twigs, the tips of which had been broken off, were also examined. In the tissue bordering the living wood the ducts were filled with pale brownish gum.

As was mentioned under "Temperature Relations" gum was also formed in abscised twigs when set in a vessel containing water and placed in the incubator.

Oak, Quercus coccinea & Q. stellata.

Gum in oak wood was first noted in twig cankers produced by inoculating with *Lasiodiplodia triflorae*. In the tissue surrounding that infected with the fungus, a dark gum like substance was deposited in comparatively small amounts. It seemed slightly less soluble in water than that in newly infected wood of plum and gave a black color with ferrous sulphate; but otherwise had all appearances of gum. Later examinations of older cankers showed gum of the typical brownish color filling wood parenchyma cells and some ducts.

Tyloses were also found in the ducts.

On September 16, 1916, three twigs each of *Q. coccinea* and *Q. stellata* were treated with copper sulphate and three of each with mercuric chloride. The two species reacted very similarly. Less tissue was killed in the oaks than in any other plants treated with these two poisons, and the poisoned tissue was very unevenly distributed. Instead of a single dead area surrounding the wound only a small amount of wood was affected here. The poison spread from this area through the cambial region and produced other small areas of dead wood at various points in the twigs. Gum was deposited fairly abundantly in the wood parenchyma and fiber cells in and around these dead areas, but little could be found in the vessels. The larger vessels were closely packed with tyloses. Occasionally gum could be found entirely filling a small vessel and usually no tyloses could be found in these. Evidently the tyloses are formed sooner than gum in such tissues and as the pits in the vessel walls are plugged by the tyloses, gum cannot pass into the vessels. It would seem from these results that tyloses as found in oak are more effective in preventing spread of poisons than are the gums, probably because they are formed immediately on disturbance of the tensions in the water column of the vessels, while gum formation is a slower process.

Liriodendron.

On October 4, 1916, three branches (about one inch thick) of thrifty young tulip poplar trees were treated with copper sulphate. The poison spread rapidly and after a few days nearly

all the wood above the wound was blackened. Very little gum could be found. Occasionally a little could be found near the edge of dead tissue but not sufficient to interfere at all with spread of the poison. This was probably due to the small number of parenchyma cells (wood of thrifty *Liriodendron* being made up almost entirely of vessels) and also possibly to the small amount of pectin substance deposited in the wall.

In examining a canker of unknown origin from a slow growing branch of an old tree, gum was found deposited quite abundantly in the border between dead and living wood.

Pine (P. strobus).

October 4, 1916, three young pine trees were treated with copper sulphate one and a half to three feet below the top. The results were much the same as those obtained with tulip poplar. The poison continued to spread for a long time until nearly all the wood of the main stem and of the branches above the wound was dead. A little gum-like substance, insoluble in alcohol and in ether, could be found occasionally. No resin was deposited until sometime after treatment. The resin ducts apparently furnished a ready means for spreading the poison. Under the microscope the blue stain of copper sulphate could be seen in the cells lining the resin ducts.

On an older tree several young twigs that had been killed by twig borers were killed and washed through several changes of alcohol, then treated with ether for several hours and embedded and sectioned. The cells of the region between living and dead wood tissue were still partially filled with a substance which stained like the gum of *Prunus* wood.

Asparagus Officinale.

An asparagus stem into which an insect had bored became infected and an area of tissue six inches long along one side of the stem was finally killed. In the bundles around and throughout this dead tissue gum of the usual light brown color was found.

Later six asparagus stems were slit, a small amount of copper sulphate inserted and the slit covered with grafting wax. As in the *Liriodrendron* and pine, the poison continued to spread for about two weeks. All but two of the tops of the plants were finally killed. In those not killed within the first few days some gum was formed and in those not entirely killed some of the bundles showed heavy deposits in the ducts and surrounding cells and some in the bundle sheath. In a stem where each bundle is separate one could of course not expect to find the well defined gum area such as occurs in the woody stems. Little or no gum is found in the non-pitted parenchyma cells and the poison continues to spread slowly through them.

Lettuce.

During the spring of 1916 some lettuce growing on the green house bench was severely attacked, when nearly mature, by some root-rot organism apparently *Rhizoctonia*. The cortex was blackened and decayed often above the first leaves and some of the plants were killed outright. The ducts in the diseased region and for some distance up into the healthy tissue were filled with gum. The gum had the usual light brown color and reacted with stains as in other plants studied. None could be found in the parenchyma cells or in any elements of the cortex.

Peanut.

In the mature peanut stem the pith rays are comparatively narrow and the woody cylinder almost complete. The cortical region is therefore more sharply set off than in most herbaceous plants. When the stem is attacked by *Cercospora personata* (B. & C.) Ellis, the mycelium of the fungus, is usually confined to the outer cortex, but occasionally a more deep-seated spot occurs and the cortical tissue is killed to the cambium. In such cases gum is deposited in the young wood cells of the cambial region and also in the nearby wood vessels.

Cotton.

During May and June of 1916 every cotton plant over one corner of an acre plot was attacked by fusarium wilt. Some of the plants were killed outright; but others sent out a branch from near the ground, which grew up and produced some cotton. When the stem of such a regenerated stem was sectioned the new growth was usually found bright and healthy. In the diseased portion there was a considerable deposit of gum in the wood parenchyma cells surrounding the infected ducts and some ducts were filled with the brownish deposit. In killing and desilicifying tissue for sectioning nearly all of the gum was dissolved out. Tyloses were fairly abundant in the infected ducts.

Tomato.

In wilt infected tomato plants conditions were found very similar to those just described for cotton. Some partially resistant plants growing in infected soil were partly killed during early summer, but during August new branches were sent out from a little healthy tissue near the surface of the soil. These new branches in most cases remained healthy until the end of the season and produced fruit. In other plants one of the original branches remained free of infection after the rest of the top was dead. In such plants the infected ducts were usually surrounded by parenchyma cells which were filled with a gum

deposit. Usually only the cells joining the infected duct form gum; but, except where this vessel comes in contact with another, this layer seems to be sufficient to hold the fungus mycelium in check. Tyloses were also quite abundant.

These observations indicate that the gum deposited in the cells surrounding infected ducts, with the tyloses and gum in the ducts, is sufficient to prevent spread of the mycelium, and that they are probably the determining factors in the partial resistance of some plants. Observation with cotton wilt has not been so extensive, as with tomato, but so far no tomato plants tried resisted infection. The mycelium merely spreads more readily in plants of some varieties than in those of such varities as Duke of York, Stone, Globe and one or two others. We hope at an early date to determine this relationship with more certainty.

Lespedeza.

A shrubby perennial species of Lespedeza from Asia was planted along a hedge at the end of a field. In plowing the fields horses had been allowed to bite off the ends of many branches. The end of the remaining twig stumps died back one to three centimeters. At the juncture of this dead portion with the living tissue gum was formed and deposited mostly in the vessels. In the wood of the plants the ducts are comparatively large and scattered, being separated by considerable quantities of small-celled woody parenchyma tissue. Very little gum was found in the parenchyma tissue. The fact that most of the gum passed into the ducts may have been due to the shape of the pits between the ducts and the surrounding parenchyma cells. The killed material was very hard and difficult to section thin enough to allow a careful study of its structure.

Some twig cankers apparently produced by an unidentified fungus were also examined. In these very little gum was found in the infected area; but in the surrounding tissue it was quite abundant.

Grape. Vitis labrusca and V. rotundifolia.

Black-rot Cankers. The fungus causing black rot of grapes (*Guignardia Bidwellii*) often attacks the leaves and young twigs of both scuppernong and the bunch varieties of grapes. Like *Cercosporo personata* on peanut and *Phyllosticta solitaria* on apple, this fungus seems to spread rather slowly and to be confined to parenchyma tissue. The mycelium was never found in wood tissue; but often the bark is killed down to the cambium. In this case gum is deposited in the young wood cells and vessels just beneath the cambium. The gum seemed to be quite soluble and much of it was lost during desilicification and washing.

Black Gum. Nyssa biflora Walt.

Some vigorous young sprouts from a broken root of this species were bitten off by cattle during the second summer of their growth. The bruised ends of the stubs died back a little and between this dead and the living tissue beneath was an area densely filled with tyloses and gum. The tyloses were most abundant toward the outer portion of this shield tissue while gum was most abundant nearer the living cells. The tissue was killed deeper around the pith of the twig, so that a cross section through the upper end of the living bark and cambium showed three areas—the living bark and new healthy wood outside, then the area densely filled with gum, and finally that next the pith with ducts filled mostly with tyloses.

Liquidambar styrasaflua L.

In a certain wooded area near the Station grounds the twigs of Liquidambar were quite commonly affected with a canker apparently produced by a fungus, the identity of which was not determined. Probably it should be referred to the genus *Cytospora*. The fungus mycelium enters the wood and continues to live and grow at least until the canker is finally overgrown. In the infested area considerable gum was found in the wood parenchyma cells which are quite abundant in this wood, but few vessels contained gum. In the shield tissue, however, all elements, wood parenchyma, wood fibers and vessels, were densely filled with gum. Rows of gum pockets extended more than half way round the twig, at what was the cambial region at the time of infection. As the new tissue extended out over the edge of the canker, it often became infected apparently from the point of contact with the dead bark, and a new row of gum pockets would be formed around the cambial region (fig. 15).

At the same time infected bark was separated from healthy bark by formation of a cork layer. Often several successive layers would be cut off and partly freed, forming a scaly protuberance on the twig.

European Hazelnut. (Corylus avellana L.)

Some years ago several clumps of European hazelnut were planted in nut tree test plats on the Station grounds. Recently they have been overgrown and shaded by chestnut and other large growing trees and many of the branches have become diseased and some have been killed by a canker produced apparently by *Endothia gyrosa*. The cankers were large, though the fungus seemed to spread very slowly after the initial attack. One branch about five inches thick, on which a canker had been developing during several seasons was cut down and some of the cankered portion sawed into thin disks for study.

About three-fourths of the cross section of the trunk was dead and that portion first infected was considerably decayed. Between this decaying wood and that still living was a dark brown layer about one-half to one inch thick in which all elements of the wood were filled with gum (figs. 3 and 14). Tyloses were also quite abundant in the wood vessels. This shield layer evidently served to hold the fungus in check. Along one edge the fungus had not been able to advance during the three previous seasons; but along the other the fungus had broken through the barrier (probably around a branch) and had spread a considerable distance before a new shield layer was formed.

Juglans Sieboldiana.

The Japanese walnut is in this region very subject to a serious trunk rot, possibly produced by *Schizophyllum Commune*. The tops of some of the trees are almost entirely killed and the inside of the trunk decayed, leaving a mere shell one-half to an inch thick. In all cases examined, whether in the old trunk or in the younger branches, a shield layer of black gum-filled tissue separated the living from the diseased wood. The gum is deposited mostly in the wood parenchyma cells, though occasionally a vessel was entirely filled. The ducts are very large and contain tyloses in the diseased region.

Maple (Acer rubrum.)

An old maple stump twelve inches in diameter and almost entirely destroyed by a heart rot was found on the Station grounds. The top had broken off about fifteen feet above the ground but the thin shell of living wood of the stump still supported a few vigorous young branches. This stump was cut for study. The wood was nearly all decayed, although there remained a little which was alive and healthy. This living wood was separated from the decayed by a dark brown gum-filled area one to five millimeters thick (fig. 4). Within this area the lumen of practically every fiber and wood parenchyma cell and every vessel was filled with the brownish gum. The comparatively large masses of gum in the vessels seemed to disintegrate very slowly. Most of them remained until the wood was well decayed. Several old shield layers which had in some way been overrun by the decay organism were still evident as black lines in the decayed wood; but there could be no doubt of the protective action of this shield layer.

Discussion.

Wounds and cankers on several other plants, including elm, sycamore, hickory, sassafras, elder, poplar, rose, beggar weed,

and golden rod, have been casually examined for the presence of gum, and some gum has been found in all cases.

It seems, therefore, that the results herein reported together with those of earlier workers in this field should be sufficient to show the general occurrence of the phenomenon of gummosis. In fact from the work of Mangin[15] and Gruss[10] we should expect to find some pectic substances and, therefore, under certain conditions, some gum formation in all woody plants.

Mangin[15] by staining reactions demonstrated the presence of pectin in the cell walls of all the great groups of plants and expressed the belief that it formed the principal constituent of the primary lamellae of all plant tissue.

In a publication the following year Gruss[10] reported the general prevalence of pectin in the cell walls of plants. He also found hemicelluloses laid down as thickenings inside the cells of thickened root stocks and of the twigs of many plants. Such thickenings as referred to by Gruss are very noticeable in the fiber and wood parenchyma cells of apple twigs. The inner wall layers of these cells give the same staining reactions as the middle lamellae, but they fail to react thus after gum is deposited in the lumen. The lumen appears also to be enlarged by gum formation, and there seems to be little doubt that the pectic substances deposited here, as well as those of the middle lamella, are transformed into gum.

It seems, however, that the greater part of the gum is formed from the middle lamella. If during the early stages of gum formation the diseased wood is carefully sectioned, the gum can be seen exuded in microscopic droplets over the pits in the cell walls (text figs. 1 and 2). The starch apparently never plays a very important part in gum formation. The fact that it sometimes disappears from cells where gum is forming probably only indicates that the same stimulus which initiated gum formation has also activated the diastase in these cells, since experiments on the relation of moisture to gummosis showed a decided correlation of this phenomenon to growth, in that a stimulus was necessary to initiate bud or callus growth in the cherry twigs.

There seems to be little possibility of doubt that the liquifaction of the pectins is brought about by enzyme activity; but our knowledge of enzymes will scarcely justify a complete explanation of the process. It seems evident, however, that the enzyme is not ordinarily present in quantities sufficient to produce evident gummosis. When the stored pectic substances in fleshy roots, in twigs, etc., are dissolved and removed, the action is slow enough to allow complete solution and removal of the jelly as fast as formed. It is only when an abnormal stimulus causes rapid formation of enzyme from its mother substance or zymogen that the pectins are broken down in such quantity as to cause gumming. That the enzyme is not constantly present in the

cell, escaping and attacking the cell walls when the plasma membrane is destroyed by wounding, etc., as suggested by Beijerinck[1], is shown by these experiments on moisture relation. Were this the case, gum would be formed most readily and abundantly on those wounds in a saturated atmosphere where the moisture would favor enzyme action. But we find exactly the opposite occurring—that is, no gum at all is formed on twigs in a saturated or nearly saturated atmosphere.

Summary.

Gum formation, although affected to some extent by variations in temperature, moisture, etc., as are growth phenomena, is not dependent upon, or always associated with, growth activity. This behavior together with the fact that a pectin dissolving enzyme is found in freshly exuded gum indicates quite conclusively that it is brought about by enzyme activity.

Apparently the mother substance or zymogen is constantly present and the enzyme is formed normally in small quantities at certain seasons; but it is only under the influence of certain stimuli that the enzyme is formed in quantities and liquifies the pectic substances faster than they can be removed, when they are deposited in the tissue or exuded as a gummy mass.

The occurrence of the phenomenon is quite general in woody plants. It probably occurs to some extent in the woody tissue of all vascular plants; and, as a means by which plants are protected from serious injury through surface wounds or fungus attacks, is of considerable importance though often entirely overlooked by plant pathologists.

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Explanation of Plates.

Plate 1.—Fig. 1. Gummosis in young plum tree, following inoculation with *Lasidiplodia triflorae*—inoculated Jan. 5, 1916, photographed July 4, 1916, after four days of rainy, damp weather.

Plate 2.—Fig. 2. Cross section of a fifteen year old apple branch infected through fire blight canker, showing dark brown layer of gum-filled tissue between healthy and diseased wood. ½ nat. size. Fig. 3. Cross section of a canker produced by *Endothia gyrosa* on a trunk of *Corylus avellana*, showing thick layer of gummous tissue between dead and living wood. 2-3 nat. size. Fig. 4. Cross section of decaying trunk of maple (*Acer rubrum*), showing very thin layer of gum filled tissue between dead and living wood. 2-7 nat. size.

Plate 3.—Fig. 5. Cross section (photomicrograph) of peach twig 42 days after treating with mercuric chloride, showing gum in wood between living and dead portions and gum pockets around cambial region. X 12 diam. Fig. 6. Cross section (photomicrograph) of apple twig 22 days after treating with mercuric chloride, showing thin layer of gum-filled wood surrounding dead area. X 10 diam. Fig. 7. Longitudinal section (photomicrograph) of blighted pear twig at juncture between diseased and sound tissue, showing tissue partly destroyed by gummy degeneration. Some gum also shows in the cells and vessels of the sound tissue. X 9 diam.

Plate 4.—Fig. 8. Cross section (photomicrograph) of *Phyllosticta solitaria* canker on apple twig, showing isolation or infected bark by cork and also gum in the wood where the fungus reached the cambium. X 11 diam. Fig. 9. Portion of a similar section more highly enlarged to show distribution of gum just beneath the cambial region. X 30 diam. Fig. 10. Cross section (photomicrograph) through a *Sphaeropsis* canker on apple twig, showing pycnidia of fungus, formation of cork and dense deposit of gum between diseased and healthy tissue. X 10 diam.

Plate 5.—Fig. 11. Portion of section shown in fig. 10 further enlarged to show dense gum deposit in border of diseased tissue while the infected area is fairly free from gum. X 35 diam. Fig. 12. Portion of cross section of black gum (*Nyssa biflora Walt*) twig just below where it had been bitten off by cattle, showing new callus tissue on the outside, then normal healthy wood, layer densely filled with gum, and further toward the pith an area with little gum but many tyloses in vessels. X 45 diam. Fig. 13. Cross section (photomicrograph), showing gum in vessels of blackened area of maple shown in fig. 4. X 35 diam.

Plate 6.—Fig. 14. Cross section (photomicograph) of *Corylus* wood from border of diseased wood, showing at right cells and vessels filled with gum and tyloses, healthy tissue at left. X 30 diam. Fig. 15. Portion of cross section (photomicrograph) of cankered twig of *Liquidambar*, showing dense deposit of gum surrounding infected area and two rows of gum pockets formed by reinfection on succeeding years. X diam.

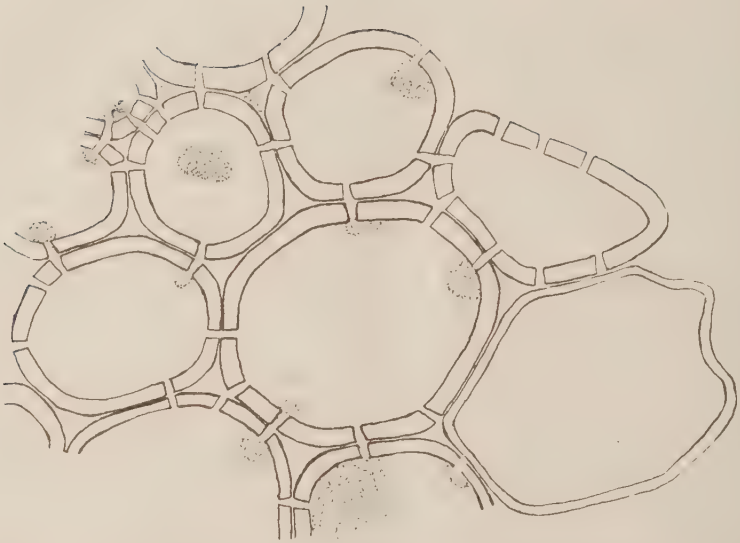

Fig. 1. Cells from the bundle sheath of peach twig treated with H mercuric chloride. Gum had begun to form and pass in through the pits before the cells were finally killed by the poison.

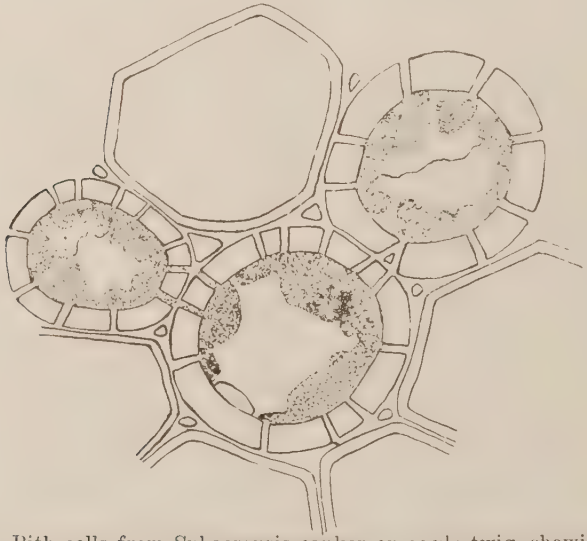

Fig. 2. Pith cells from Sphaeropsis canker on apple twig, showing gum entering lumen of conducting parenchyma cells through pits.

PLATE NO. 1.

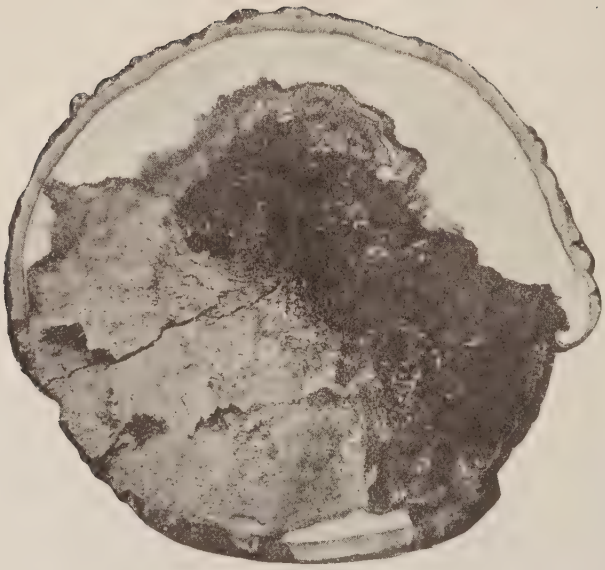

Fig. 2.

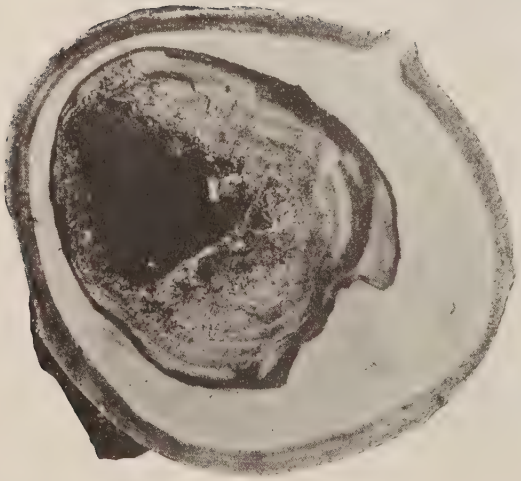

Fig. 3.

Fig. 4.

PLATE NO. 2.

Fig. 5.

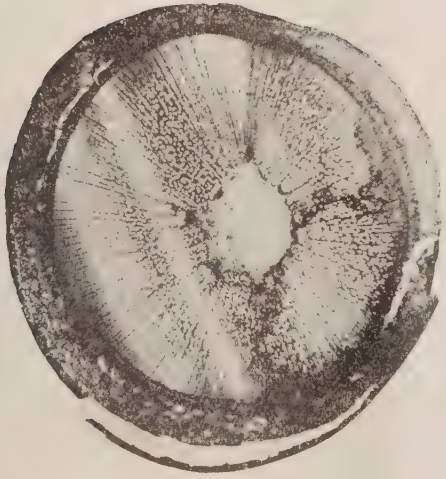

Fig. 6.

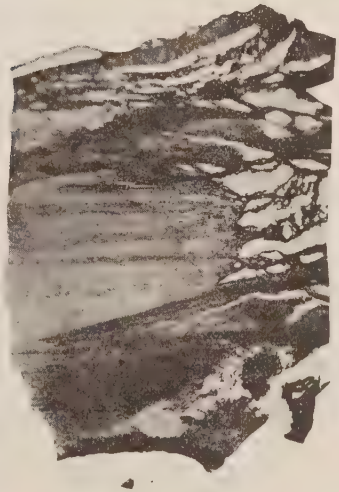

Fig. 7.

PLATE NO. 3.

Fig. 8.

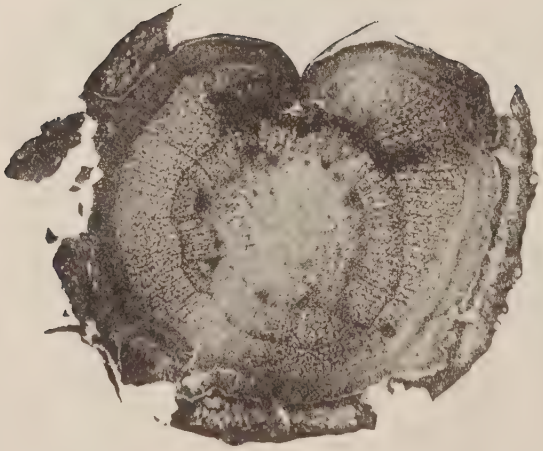

Fig. 9.

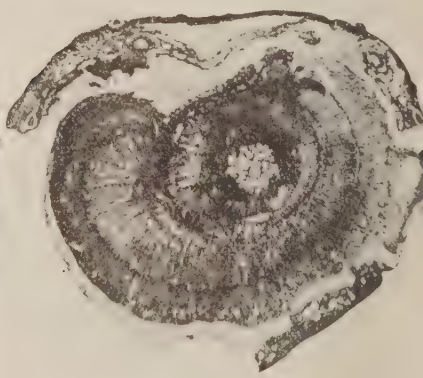

Fig. 10.

PLATE NO. 4.

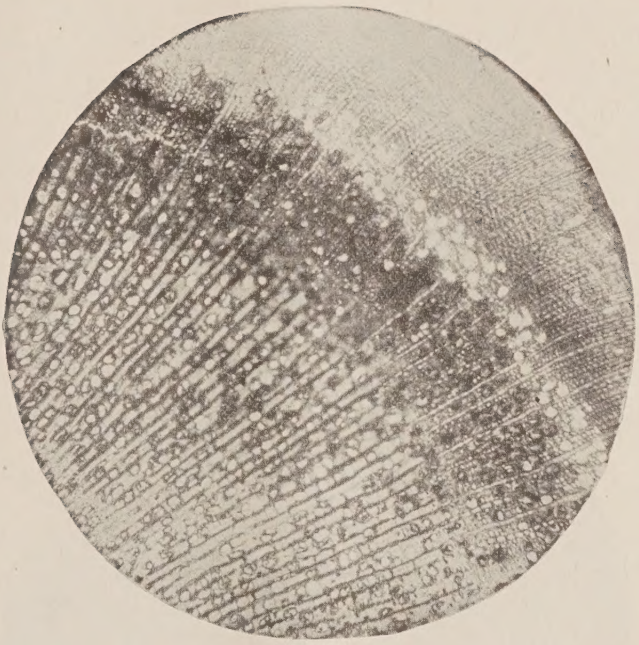

Fig. 11.

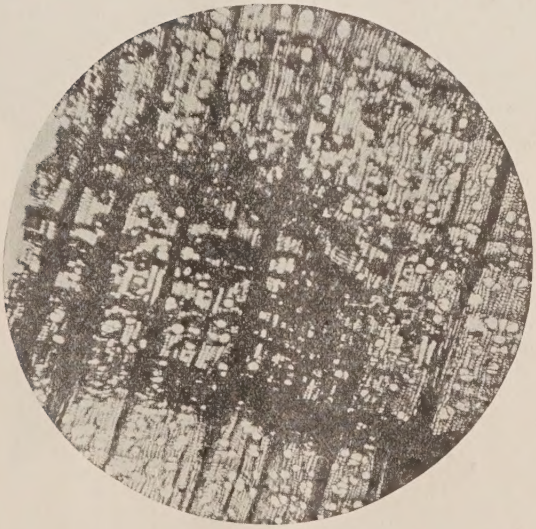

Fig. 12.

PLATE NO. 5.

Fig. 13.

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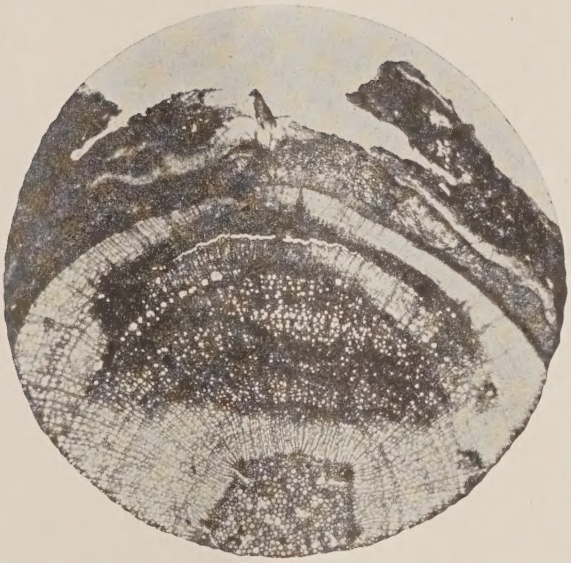

Fig. 14.

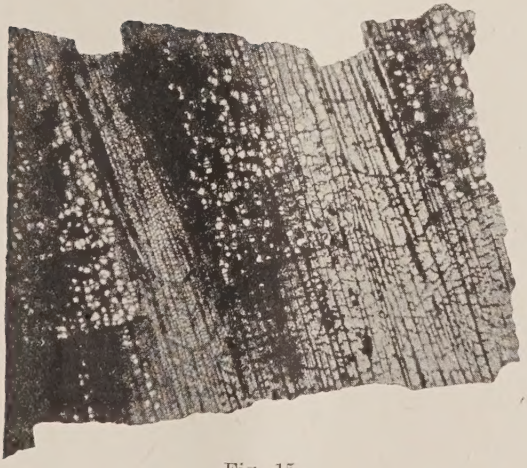

Fig. 15.

PLATE NO. 6.